

LETTERS TO THE EDITOR

Laser Angioplasty and Atherosclerosis

The assertion of Isner and Clarke (1) "without equivocation" that "laser-generated light *eliminates* atherosclerotic plaque" is not true, if by "eliminate" they mean "remove completely." On the contrary, lasers have merely been demonstrated to bore a hole in a plaque by vaporizing material in the path of the beam, which is quite different from eliminating the plaque. What they describe as laser angioplasty is actually a form of incomplete endarterectomy.

It is important to remember that atherosclerosis is *not* simply a deposit within the vessel wall, but a transformation of the intima into an atherosclerotic plaque, with alteration of the media (2). Endarterectomy, which is necessarily incomplete when performed by laser, does not leave behind a normal vessel. It is unrealistic to think of local vaporization of a portion of a plaque as elimination of the plaque. Moreover, atherosclerosis is a diffuse disease and is not confined to the area of worst narrowing as seen on angiography (3).

Based on the poor long-term results of local (mechanical) endarterectomy *without* bypass (4), there is no reason to assume that the results of incomplete local endarterectomy performed by laser should be any better. It is a mistake to think that lasers have the power to make atherosclerotic lesions magically disappear.

It may be that the Gordian knot cannot be unraveled: Once structural changes take place in the vessel wall, it may be impossible to return that vessel to its predisease state. As in so many diseases, the only true "cure" lies in prevention.

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References

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3. Arnett EN, Isner JM, Redwood DR, et al. Coronary artery narrowing in coronary heart disease: comparison of cineangiographic and necropsy findings. *Ann Intern Med* 1979;91:350-6.
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Reply

As Weinstein correctly points out, we did not intend to imply that clinical application of laser irradiation is likely to restore an atherosclerotic coronary artery to its virgin, nonpathologic state. As he, and we (his Ref. 2), have indicated, the fact that the atherosclerotic process permanently alters the normal architecture of the coronary wall (by "eliminating" a substantial portion of medial smooth muscle) implies that restoration of a truly normal coronary artery is an unrealistic end point.

In contrast to alternative, so-called definitive interventions, however, laser irradiation does, in fact, "eliminate" atherosclerotic plaque: rather than a stenosis that persists unchanged after bypass surgery, or an atherosclerotic lesion that persists in remodeled form after balloon angioplasty, a substantial portion of atherosclerotic plaque may be effectively debulked by laser irradiation.

Weinstein is also correct in pointing out that laser ablation of atherosclerotic plaque is similar in many respects to mechanical endarterectomy, except, of course, that laser irradiation offers the potential of a percutaneous, as opposed to open chest, procedure. Whether the clinical results that can be accomplished using laser irradiation will be superior to those previously achieved by mechanical endarterectomy is admittedly a critical question that must be resolved by animal or human investigations, or both.

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Manipulating the aT-eT

O'Donnell et al. (1) found that the aT-eT interval (time from the apex to the end of the T wave) was significantly shorter at maximal or near-maximal heart rates in the groups with than in the groups without coronary artery disease. They used another index, T slope/T-amplitude (ratio of the maximal slope of the descending limb of the T wave to its amplitude), and found a similar separation. On closer analysis, however, it is apparent that this more complicated index is not materially different from aT-eT, and indeed is virtually its reciprocal.

Using their Figure 1, it can be seen that the descending limb of the T wave is approximately linear so that its second part is almost triangular. A reasonably accurate estimate of T slope is then $(-)\text{T amplitude}/\text{aT-eT}$. The index of O'Donnell et al., which is T slope divided by T amplitude, is thus merely $(-)\text{1/aT-eT}$. Predictably, during exercise this index increases numerically but negatively as aT-eT decreases in the groups with disease (their Fig. 2). To be sure, other variables, such as a less sharp T wave apex, or a curved T wave descent, affect the validity of approximating the index to $(-)\text{1/aT-eT}$. That these variables compared with aT-eT are minor can be deduced from the observation that the product of aT-eT and the corresponding index (obtained from their Tables 3 and 4) is fairly constant. Multiplying aT-eT by the index, its reciprocal, eliminates it so that the constancy of the resultant product implies that other variables are relatively unimportant.

The authors concluded that "there is considerable overlap in the individual data" for aT-eT so that its sensitivity and specificity as a marker of disease were impaired. However, while the absolute